CHAPTER 16

Neuropsychological Assessment in Posttraumatic Stress Disorder

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INTRODUCTION

Reviewing and discussing the neuropsychological assessment of posttraumatic stress disorder (PTSD) is a challenge for a number of reasons. As a recent inclusion in the ***Diagnostic and Statistical Manual of Mental Disorders*** (i.e., appearing in DSM-III; American Psychiatric Association, 1980), comparatively little has been written to date on PTSD from a neuropsychological perspective. Major emphases early in the development of the PTSD literature centered around defining the set of primary PTSD criteria and associated features, debating the validity of its status as a unique anxiety disorder, developing reliable diagnostic interviews and psychological methods for measuring PTSD, expanding conceptualizations of the role of trauma in the production of PTSD in various populations, and exploring psychological and pharmacological treatments. The PTSD criteria have remained relatively stable across revisions of the DSM, and diagnostic interviews for PTSD have been created to classify cases in clinical and research protocols. Over the past 15 years, developments in these areas have established a firm foundation for now focusing on etiological questions.

Another reason for the relative dearth of formal neuropsychological study of PTSD may be that, at this point in time, few clinical neuropsychologists have been trained directly to study trauma, and few traumatologists are trained in the systematic study of brain–behavior relationships. Clinical neuropsychologists have traditionally focused on the evaluation and treatment of identifiable neurological conditions of the central nervous system that produced disruptions in cognitive functioning and whose presence could be corroborated by other diagnostic methods, for example, autopsy, electroencephalogram (EEG), angiography, computed tomography (CT) scan, magnetic resonance imaging (MRI), serum assays, and cerebrospinal fluid (CSF) analysis. Psychiatric disorders were not as readily characterized by these

methods, and psychiatric conditions, historically, might have been invoked to explain symptoms when no classic neurological explanation emerged. Psychiatric disorders now represent an ever increasing portion of the neuropsychological literature as the roots of disordered emotion, behavior, and information-processing are conceptualized from a brain-based, biological–psychiatric perspective. Clinical neuropsychology has long been the nexus between brain function and dysfunction, affect, and behavior and cognition; and it will continue in this role for future study of trauma effects on the brain.

Considering the potential physical and neurological damage that often accompanies psychologically traumatic events, relatively little of the trauma literature addresses these aspects in depth. Traumatologists as a group view PTSD largely from either the perspective of psychological constructs indexed to behavioral observations, self-reports, and psychophysiological responding, or from biopsychological perspectives indexed to psychopharmacological studies. Explanations for PTSD symptom development, maintenance, alteration, and remission involve mainly operant and classical conditioning processes, psychodynamic formulations, and models of neurotransmitter system functioning. Traumatologists and clinical neuropsychologists would most likely agree in principle that familiarity with each others' perspectives is important for understanding the complexities of PTSD, but cross-reading two extensive literatures has practical limits, and applying the synthesis of these perspectives to actual cases or research designs is a larger challenge needing a framework and guidance.

To facilitate the synthesis and application process, this chapter will focus on the status of neuropsychological contributions to understanding the neurocognitive concomitants of PTSD symptoms. The main goals are (1)to introduce trauma clinicians and researchers to brain-based views that are important to consider in their work, and (2) to familiarize neuropsychologists with PTSD-related factors that may not be typically considered or addressed during evaluations and case formulations. Content sections of this chapter will (1)review the relevant literature on neuropsychological evaluation to date, (2) present general assessment issues related to trauma populations and specific considerations in conducting neuropsychological evaluations with PTSD patients, and **(3)** conclude with a discussion of future directions and issues of central concern given the emergent status of empirical research and model development in this area.

BRAIN-BASED CORRELATES OF PTSD

The degree to which PTSD coexists with, is caused by, or is the cause of neurocognitive problems is a critical issue needing conceptualization and systematic study. At issue is not just the task of specifying additional methods to identify cases of PTSD, but also the pursuit of variables that inform us about why PTSD develops in some trauma-exposed individuals

and not others, as well as the factors that modulate the severity of symptom manifestation following exposure to trauma. The findings from the National Vietnam Veterans Readjustment Study (NVVRS; Kulka et al., 1990) suggest that alternatives in addition to traumatic exposure need to be considered as determinants of PTSD.

The NWRS found prevalence rates of 30.9% for male combat veterans meeting diagnostic criteria for lifetime PTSD, and another 22.5% who developed partial PTSD after the military. Of these, 15.2% had symptoms at levels sufficient to meet the requirements for current PTSD at the time of the study. Comparisons among prevalence rates for lifetime and current PTSD show that about 49% of the male theater veterans who ever exhibited symptoms continued to have symptoms, supporting the contention that PTSD is a chronic disorder. The overall rates were higher among Black and Hispanic veterans and among those experiencing heavy combat. From an absolute perspective, the current PTSD group is a large number of afflicted individuals, but it is a relative minority of the sample.

Various psychosocial factors from the NVVRS were analyzed to investigate potential predisposing factors. Statistical adjustment for predisposing factors had the most impact on reducing prevalence rates for combat theater veterans. The variables found to contribute consistently to the predisposition adjustment models were having drug abuse or dependence before the military, being raised in a family that had a hard time making ends meet, having had symptoms of an affective disorder before going to Vietnam, and exhibiting problem behaviors in childhood. Although not an established relationship in the NWRS, these variables could reflect patterns of adjustment to preexisting trauma, developmental conditions such as learning disabilities and attention deficit disorder, or other neurological problems. It may also be the case that a coexisting neurobiological condition contributes to the development and maintenance of chronic PTSD symptoms in this minority portion of the sample. A role for nontrauma exposure variables is suggested by structural equation modeling of the NVVRS data conducted by King and King (1995), who concluded that the effects of combat exposure were mediated by the outcome of cognitive processing of perceived threat. Schlenger (personal communication, 1995) found a correlation of .24 between the continuous version of the NWRS global war zone stress exposure index and the probability of developing PTSD for Vietnam theater veterans. The small amount of variance attributable to this relationship combined with the minority percentage prevalence rates suggests that those who develop chronic PTSD may have a particular response to trauma possibly based on constitutional variables. To advance beyond psychological and diagnostic studies of PTSD, neuropsychological aspects of constitutional variables should be investigated by trauma researchers for their causal relationship to clinical symptom development and maintenance.

More is hypothesized and less is known about the relationship between enduring neurocognitive changes and the corresponding neurobiological

mechanisms underlying the brain's response to trauma. Although models are emerging, at present there is no well-formulated consensus model that can be used to guide neuropsychological investigations. Assessing PTSD neuropsychologically involves some a priori assumptions about the relationship between traumatic experiences and brain functioning, which also require further elaboration and refinement. In the absence of a dominant neuropsychological model to serve as a guide, hypothesized neurobiological systems, logical extensions from knowledge of related disorders, and patterns among clinical features must serve the purpose. In attempting to apply neuropsychological methods to clinical evaluations and research protocols, two considerations are of clear importance: (1) What is the relationship between the traumatic event, PTSD development, and the neurocognitive impairments observed; and (2) what underlying brain systems are related to the patterns of cognitive symptoms that manifest clinically?

As with psychiatric disorders in general, PTSD does not conform to classic neurological lesion analysis. Unlike a tumor, stroke, aneurysm, or multiple sclerosis, psychiatric disorders often have few or no corresponding neuroanatomical correlates that are pathognomonic and identifiable via autopsy, CT scan, or MRI scan (Raz, 1989). When findings are present, they may not reliably correlate with the level of functional deficits or symptom severity (Devous, 1989, p. 219). However, advances in the behavioral neurosciences, electrophysiological measurement (quantitative EEG, evoked potential mapping), and functional neuroimaging techniques, such as positive emission topography (PET), single-photon emission computed tomography (SPECT), and functional MRI, show promise as a basis for possible development of nonpsychological models of functional disorders (Andreasen, 1989; McGuire, Shah, & Murray, 1993) and can provide appropriate correlative methods for studying psychiatric disorders by noninvasively measuring metabolic functioning of the living brain as it actively processes information. Thus far, studies employing these methods have primarily focused on schizophrenic disorders and mood disorders followed by some of the anxiety disorders such as obsessive–compulsive disorder and panic disorder (Orsillo & McCaffrey, 1992). The logical extension for studying anxiety disorders is the application of these methods to PTSD.

Some recent articles have suggested neurohiological aspects of brain functioning that might he involved in the development of PTSD symptoms. Watson, Hoffman, and Wilson (1988) postulated neurochemical changes in the locus coeruleus and amygdala following exposure to uncontrolled stress. Kolh (1989) hypothesized that changes at synaptic levels of cortical neurons in PTSD patients result from protracted and excessive sensitizing stimulation, eventually producing alteration in habituation learning. Other investigators have more specifically focused on ***activation of the beta-adrenergic stress hormone systems in response to emotional experiences*** (Cahill, Prins, Weber, & McGaugh, 1994), the central role of ***abnormal noradrenergic function in the pathophysiology of PTSD*** (Krystal et al., 1989; Southwick et al., 1993); and ***altered***

functioning of brainstem catecholaminergic systems in childhood **PTSD** (Perry, 1994; cf. Charney, Deutch, Krystal, Southwick, &Davis, 1993, for a review of the psychobiological mechanisms of PTSD). Some of the neurobiological formulations are extrapolations from preclinical studies, and others follow from investigations with human subjects. These neurobiological perspectives suggest mechanisms for some PTSD symptoms but only more generally posit the impact of trauma on complex mental processes and higher cortical functioning.

Regional brain areas previously hypothesized as responsible for PTSD symptoms, including the locus coeruleus, hippocampus, and septo-amygdalar complex continue to be studied. Bremner and coinvestigators (1995) found that the mean right hippocampal volume from 26 MRI scans of combat-PTSD subjects was 8% smaller than for control subjects. No differences were found in volume for other brain regions. Using PET to measure regional brain function, Semple, Goyers, McCormick, and Morris (1993) reported regional blood flow differences between control subjects and PTSD males with substance abuse histories in the areas of the hippocampus, parietal lobes (decreased flow), and orbito-frontal complex (increased flow). Ito, Teicher, Glod, and Harper (1993) employed neuroimaging, neuropsychological, and electrophysiological methods to investigate the association between abuse histories and neurological abnormalities in 104 child and adolescent inpatients. Physically and/or sexually abused subjects showed increased abnormalities in left frontal, temporal, or anterior regions, while those experiencing primarily neglect or psychological abuse showed only left temporal abnormalities. To date, no consensus functional scanning correlates of PTSD have been established; yet, the findings in the aforementioned studies point to the presence of alterations in brain areas associated with neuropsychological deficits in other related disorders.

Various brain regions have been associated with clinical symptoms in other disorders that share features with PTSD. Disorders of arousal and attention have been generally associated with frontal system dysfunction (Stuss & Benson, 1984) and fronto-subcortical pathology (Damasio, 1985). Orsillo and McCaffrey (1992) described findings suggesting that temporal lobe epilepsy and trauma to the temporal area results in symptoms similar to those seen in anxiety disorders. Alternative models and hypotheses suggest a relationship between left frontal lobe and left temporal lobe dysfunction in panic disorder and obsessive–compulsive disorder (Volkow, Harper, & Swann, 1986). Uddo, Vasterling, Brailey, and Sutker (1993) studied memory problems in PTSD veterans and noted that investigations into the pathogenesis of PTSD implicate brain structures involved in memory, such as the limbic system and septo-hippocampal-amygdalar complex. Brende (1982) conducted a study of lateralized electrodermal responding (EDRs) in six trauma-exposed subjects, and found that (1) peripheral EDRs reflected activity of the contralateral hemisphere, (2) left hemisphere functioning was associated with hypervigilance symptoms, and (3) right hemisphere functioning was associated with

emotions and imagery. Finding multiple cortical and subcortical regions via neuroimaging protocols and other methods that are linked to symptoms is consistent with the varied clinical presentations of PTSD patients, who often also exhibit psychiatric features of depression, panic, generalized anxiety, and obsessive–compulsive disorders. Multiple, relevant brain regions may also reflect the influence of neurological conditions existing comorbidly with PTSD.

NEUROPSYCHOLOGICAL COMORBIDITY AND PTSD

The comorbidity of PTSD symptoms with those of assorted psychiatric disorders compounds the task of defining and studying the range of traumatized samples seeking treatment. As complexity of the clinical presentation increases, attributing causality to any given variable correspondingly increases in difficulty. Beyond diagnostic comorbidity, other factors influence the complex manifestations of PTSD, including the spectrum of possible traumatogenic events previously experienced by trauma survivors, the range of trauma-specific parameters determining the severity of the trauma experiences, and the individual coping responses exhibited by diverse populations exposed to trauma. Along with other neurologically related factors, these factors likewise contribute to the difficulty in studying the neuropsychological concomitants of PTSD. Unless it is assumed that all of these factors are equipotent for producing neurocognitive changes, clinical and research protocols must systematically consider their impact singularly and in concert (Merskey, 1992). The discussion that follows addresses the multiply determined clinical presentations that are more common in chronic PTSD cases.

Sources of Neuropsychogenic Trauma

Many types of potentially traumatizing experiences are found across clinical PTSD samples. For traumatic events involving physical injury, traumatologists will collect specifics about the incidents and build a model to describe the socioemotional effects of the experience on the individual and related others. Neuropsychologists will gather information about the same events and focus on neurobehavioral consequences that will impact the individual. Many events that threaten the physical integrity of the person and result in the production of PTSD symptoms also have associated effects on brain functioning. Examples of traumatizing events particularly relevant to neuropsychological formulations span a range of categories. Depending on the level of injury to the brain, either cumulative or from a single episode, all of the following could be sources of neuropsychogenic trauma:

- ***Interpersonal violence:*** beatings from family members, beatings from strangers, near-death experiences from stabbing, shootings, strangulation, rape.
- ***Motor vehicle accidents:*** as a driver, passenger, witness, or pedestrian.
- ***Natural disasters:*** physical injury from floods, flying debris in high winds, collapsing structures.
- ***Other threat to life and health:*** from falls, physical punishment, electrocution, suffocation, high-risk sports.
- ***Industrial/occupational accidents:*** falls, electrocution, fires causing anoxia, equipment malfunctions causing head injury.
- ***Military-related injuries*** (combat and noncombat):
  Head wounds: stabbings, low- and high-velocity missile wounds.
  Concussions from direct explosions: land mines, hand grenades, bombs, rockets, mortars, or from firing large-scale artillery weapons.
  Falls: from training equipment, from air transportation, trees, rocks, cliffs, bridges, buildings, moving armored vehicles, personnel carriers, or jeeps.
  Torture as prisoners of war: nutritional deprivation, beatings, electrocution.
  Exposure to neurotoxins and noxious gases.
- ***Other civilian punishment and torture conditions:*** can involve starvation (Gurvit, 1993; Sutker, Allain, & Winstead, 1987; Sutker, Galina, West, & Allain, 1990), high levels of induced pain, and electrocution (Cooper & Milroy, 1994; Daniel, Haban, Hutcherson, Botter, &Long, 1985; Hopewell, 1983; Mellen, Weedn, & Kao, 1992; Millen, 1993).

These potentially traumatic experiences carry varying degrees of risk for associated neurocognitive problems. Some of these conditions and neuropsychologically relevant variables will be discussed in greater detail.

PTSD and Developmentally Based Neurocognitive Problems

Learning disabilities and attention deficit disorder have been recently investigated as comorbid conditions with PTSD. Adults with developmental disorders, which in clinical PTSD populations may not have been previously diagnosed, carry a set of behavioral and learning problems in verbal and nonverbal modalities that can preexist or are coincident with life events that would include traumatic experiences (Gaddes, 1985; Rourke, 1985; Spreen, 1988). Learning disabilities and attention deficit disorder may occur separately or in conjunction with each other, and concurrently with anxiety and depression (Biederman, Newcorn, & Sprich, 1991; Bigler, 1990). Children with some types of behavioral problems may be at higher risk for acquired injuries. Hyperactive children in households of irritable, intolerant, and violent caretakers may be subjected to physical punishment and may engage in play that results in injuries (Cuffe, McCullough, & Pumariega, 1994;

Famularo, Kinscherff, & Fenton, 1992a). Histories of academic difficulties, and the development or exacerbation of family and social problems for these individuals, may have been noted from interview data to have a correlated onset that closely postdates an injury or follows a series of subclinical events. Although clinical correlations have been observed for these conditions, the exact role of learning disabilities and attention deficit disorder in the development and maintenance of PTSD is unknown. However. establishing the history of these conditions in patients undergoing neuropsychological evaluations is important for accurately interpreting test findings.

PTSD Resulting from Neurological Conditions and Neurological Trauma

Clinical and research protocols should include assessments of pretrauma, trauma-indexed, and posttrauma neurological factors capable of producing neuropsychological deficits. Although the presence of these factors complicates causal models and interpretation, ignoring them increases the risk of misattrihuting the sources of impaired test performances.

Traumatic Head Injury

Penetrating head injury (PHI) and closed head injury (CHI)—major and minor—can coexist with psychological trauma. Penetrating head injury constitute 2–6% of all head injuries, and can include open head wounds from knives, sharp or blunt objects; skull fractures associated with falls, beatings, crush injures; or, missile wounds from gunshots, shrapnel, and other projectiles. Military combat, crime episodes, and miscellaneous accidents are the most common sources of PHI (Kampen & Grafman, 1989). Amount of damage depends on the velocity of the object and the cavitation surrounding the track of the object. Hemorrhaging occurs locally and throughout the brain. High-velocity wounds are frequently fatal, whereas low-velocity wounds such as stabbings produce local damage. Changes in behavior, affect, and cognition will be a product of the tissue damage and the resulting alteration in regional brain metabolism. PHI will always he obvious and assessable by patient report and documented medical history; however, reports of CHI events may be more variable, especially if no treatment was received.

In severe CHI, gross acceleration–deceleration movements of the brain within the skull produce widespread diffuse damage due to axonal shearing and concussive damage to the surface of the cortex in orbito-frontal, anterior and inferior temporal regions of the brain (Davidoff, Kessler, Laihstain, & Mark, 1988; McAllister, 1992). For minor head injury, movement effects are less pronounced and result in more circumscribed deficits related to abilities mediated by the anterior cortex, such as memory, attention and concentration, judgment, and abstract reasoning (Barth et al., 1983; Bigler & Synder, 1995; Kwentus, Hart, Peck, & Kornstein, 1985). Even in cases of com-

mon whiplash injuries in which no head contact or loss of consciousness is reported, complex attentional processing can be impaired (Radanov, Stefano, Schnidrig, Sturzenegger, & Augustiny, 1993).

Segalowitz, Lawson, and Berge (1993) reported an incident rate of 30% for head injuries occurring in a general population of 3,961 people sampled from high schools, university psychology classes, and general physicians offices. Reporting a head injury was significantly related to other reports of hyperactivity, sleep disturbance, depression, and problems with social functioning. Levin (1985) noted that in some patients with minor CHI, residual deficits might be manifested only under conditions of stress. Neurobehavioral effects of CHI can overlap with symptoms of anxiety, depression, manic behavior, or organic personality disorder (McAllister, 1992), and confound the differentiation between associated features of PTSD. Rattok and Ross (1993) assessed for PTSD in a traumatically head-injured population and found that 18% met criteria for PTSD. The PTSD symptoms existed independently of cognitive deficits.

Although some sources of traumatic psychological stress mentioned previously are capable of producing multiple neurological problems, the following need to be considered in particular for their concomitant head-injury potential: random physical assaults (Shepard, Quercohi, & Preston, 1990), torture (Rassmussen, 1990), domestic violence, falls from high places, sports and sports accidents (Roberts, Allsop, & Barton, 1990), industrial accidents, domestic accidents, airplane accidents, and motor vehicle accidents (MVAs).

For MVAs, a wide range of head and body injuries can be sustained by drivers, passengers, and pedestrians, with attendant psychological adjustment problems for loss of function from residual chronic symptoms (Kuch, Evans, &Watson, 1991). When the effects of the injury are combined with the symptoms of PTSD from the life-threatening event and mood disorder secondary to the PTSD, the task of attributing the relative contribution of any given factor to the cognitive changes reported by the patient and formulating a treatment plan becomes more difficult. Horton (1993) reported a case study of concurrent PTSD and head injury following a MVA that illustrates the point. As part of a multicar accident, the patient witnessed the death of the driver of another car, who fell asleep at the wheel, producing the crash that demolished the patient's car. PTSD symptoms and cognitive problems were reported. Within 3 months of treatment with behavioral techniques, the PTSD symptoms abated, but memory problems at work remained. Neuropsychological testing found mild to moderate impairments in pattern consistent with mild CHI. The treatment approach was augmented as a result of the test findings to include instruction in compensatory memory methods and organizational strategies.

PTSD can develop in cases of head trauma when no memory or only sparse memory exists for the event. A multisystems theory of memory was advanced by Layton and Wardi-Zonna (1994) to account for PTSD reactions in patients with head injuries and no recall of the traumatic accident that

caused their injury. Two cases of PTSD with concurrent neurogenic amnesia for the traumatic event were presented to illustrate the dilemma of assigning a diagnosis in the absence of conscious memory for the experience. They argued against the notion that neurogenic amnesia functions protectively to prevent posttraumatic emotional reactions. Aspects of the traumatic experience appear to become sufficiently encoded to produce reexperiencing symptoms.

Watson (1990) notes that symptoms of head injury and PTSD overlap in two cases of MVA survivors who suffered severe concussive injuries producing amnesia for the event. Reexperiencing of the event took the form of specific pain and other physical symptoms. Baggaley and Rose (1990) also reported a clinical case involving a soldier who was in a vehicle that exploded, killing eight other passengers. Coma lasted 36 hours, with a posttraumatic amnesia lasting 72 hours. While recovering physically, he developed PTSD without intrusive recall, and with nightmares characterized by unretrievable content. The phobic avoidance was consistent with specific trauma cues and was treated successfully with in *vivo* exposure methods.

McMillan (1991) described a case of severe head injury, amnesia for the event, and PTSD following a traffic accident. Despite absence of memory for the trauma, the patient experienced intrusive thoughts about the other passenger who had died, as well as avoidance of cognitive and physical events associated with consequences of the accident. Neuropsychological testing results (test battery not reported) were interpreted as revealing a moderate degree of general impairment that improved modestly on repeat testing 7 months later. Treatment using behavioral intervention was successful. PTSD clearly developed, even with loss of consciousness and organic amnesia for the event and its immediate consequences. The direct sequelae of the traumatic event and the head injury can each produce disruptions to interpersonal and intrapersonal domains of functioning for the trauma survivor.

Neurotoxic Exposure

Neurotoxic exposure can accompany traumatic events that occurred in military combat, industrial accidents, environmental accidents, suicide attempts, and intentional or accidental poisonings. The negative effects of toxin exposure on neurocognitive abilities, affect, and personality have been well described (Hartman, 1988, 1992; White, Feldman, & Proctor, 1992). Schottenfeld and Cullen (1985) reported finding that PTSD developed frequently in workers who were acutely or chronically exposed to toxins.

Hypoxic/Anoxia Episodes

Psychological traumas involving suicide attempts via carbon monoxide poisoning, respiratory-suppressing drug overdoses, near drownings (accidents, floods, storms), fires, chest injuries affecting circulation (crush injuries, stab wounds, shooting wounds), toxic spills and acute exposures, and elec-

trocution can produce neurological damage that may have an acute and chronic impact on cognitive abilities. A variety of neuropsychological deficits have been noted to occur from the nonspecific neuropathological changes following anoxia, including dysfunctional memory attributed to reductions in hippocampal volume (Hopkins, Weaver, & Kesner, 1994; Hopkins et al., 1995).

Preexisting PTSD Exacerbated by the Onset of a Neurological Condition

These patients may present with increased PTSD symptoms following the development of a neurological disorder or onset of an acute condition. The exacerbation may result from the added effects of the neurological condition to existing trauma symptoms, direct triggering of trauma content, or indirectly as a threat to health that having the disease implies. Alternatively, the disease process may diminish the patient's capabilities for suppressing negatively valenced memories and affect. Chemtob and Herriott (1994) report a case of PTSD as a sequela of severe Guillain–Barré syndrome in a 24-year-old female. Cassidy and Lyons (1992) reported on the case of a 63-year-old, World War II male combat veteran who experienced increased dissociative episodes of hand-to-hand combat, intrusive recall of traumatic memories, and avoidance of reminders of the war after surviving a cerebral vascular accident (CVA). Clinically, the disinhibiting sequelae of CVA in the previous example above have also been observed with head injury occurring at some interval posttrauma that produced increased levels of hypervigilance, daytime flashbacks, intrusive memories, with decreased control over rage episodes, and decreased ability to override persistent physical reactions after being startled.

Neurological Factors Increasing Risk for Experiencing Trauma and Developing PTSD

Having a neurological condition can affect areas of higher cognitive processing involved in learning and memory, attention, planning, reasoning, judgment, monitoring of the environment, awareness of consequences, volition, sequencing abilities, controlling impulsivity, integration of complex information, and self-regulation (Brooks, 1989; Lezak, 1989). The psychosocial impact of impairments in these areas may increase the risk of incurring a trauma. Alterations in judgment and decreased appreciation of behavioral consequences may result in various kinds of accidents, beatings, or assaults.

Neuropsychological Effects of Substance Abuse in PTSD–Chronic Alcoholism

Alcohol is frequently abused by individuals with PTSD as an agent for regulating physiological arousal, promoting sleep, decreasing pain, and numbing

responsiveness (Keane & Wolfe, 1990). Drinking occurs at varying rates and intervals. As a result, the potential neuropsychological effects are salient for PTSD patients. Harper, Kril, and Daly (1987) noted that radiological evidence indicating cortical atrophy in chronic alcoholics and in heavy social drinkers showed correlations with clinical and neuropsychological deficits. Their neuropathological studies revealed reduced brain weights and increased pericerebral space attributed to loss of white matter, particularly neuronal cell death and axonal degeneration in the anterior frontal lobe (superior frontal gyrus).

Parsons (1987) described negative cognitive effects of alcohol abuse. Neuropsychological deficits are predicted by frequency of drinking occasions and maximum quantity consumed each time. By their self-appraisal, alcoholics rate themselves as having reduced efficiency for common tasks requiring memory, problem-solving, perceptual–motor functioning, language, and communication. As duration of drinking increases, impairment scores approach those of brain-damaged patients. Much variability has been observed in the degree of deficits exhibited by alcohol abusers, as well as in recovery of function rates once abstinent. Alcohol impacts individual cognitive abilities, mood, behavior, and social functioning.

Gill and Sparadeo (1988) noted that 50–70% of all head-injury patients who were injured in MVAs had been drinking. The presence of alcohol can exacerbate the short-term neurobehavioral effects of head injury and delay or complicate recovery. Edna (1982) reported that injured patients having alcohol in the bloodstream had lower levels of consciousness when admitted and longer duration of coma not accounted for by other factors like skull fractures and hematomas. As a readily available substance that can be effective in modulating the affective states associated with PTSD, the impact of alcohol abuse on cognitive processes needs to be given serious consideration in formulations of neuropsychological status in trauma patients.

Neuropsychological Effects of Substance Abuse in PTSD–Chronic Stimulant Abuse

The effects of chronic cocaine use on neuropsychological functioning including new learning and memory, have been documented (Ardila, Roselli, & Strumwasser, 1990; Uddo & Gouvier, 1990). Hoff, Riordan, Alpert, and Volkow (1991) found impaired performances on the Wisconsin Card sorting Test and the an abbreviated Booklet Categories Test for a group of 20 cocaine abusers compared to 20 controls. Baxter and colleagues (1988) employed PET to study cerebral glucose metabolic rates of cocaine abusers in recent withdrawal. Previous studies in the literature had found decreases in anterior cortex, particularly in prefrontal and left-anterior temporal regions. However, when controlling for depression via the inclusion criteria, the findings of previous studies were not replicated. Azrin, Millsaps, Burton, and Mittenberg (1992) concluded that detrimental cognitive effects found

for cocaine abusers were significantly related to length of abstinence and were reversible within 6 months of abstinence. As cocaine has been found to be an actively abused drug of choice among clinical PTSD patients (Cottler, Compton, Mager, Spitznagel, & Janca, 1992; Famularo, Kinscherff, & Fenton, 1992b; Fullilove, Lown, & Fullilove, 1992; Hamner, 1993; MacKay, Meyerhoff, Dillon, Weiner, & Fein, 1993), current use or recent abstinence needs to be considered as a confound when interpreting test performances.

Multiple Comorbidities

In clinical settings treating chronic PTSD, many more comorbid conditions can be present (Sierles, Chen, McFarland, & Taylor, 1983), and complex manifestations may be common. For instance, it may be the case that a patient reports being in a relationship involving repeated physical battering; has a childhood history of physical and sexual abuse, possibly from multiple perpetrators; and has developed a substance abuse problem. The substance abuse problem may be severe enough to produce MVAs while intoxicated (Hillbom & Holm, 1986). In constructing the DSM-IV profile, the symptoms may meet criteria for an Axis I diagnoses of PTSD, major depression, substance abuse disorder, panic disorder, and an Axis II personality disorder. This clinical presentation can be straight forwardly conceptualized from the trauma perspective, with the clinical symptoms explained psychologically. Yet, depending on the extent and severity of head injury from the beatings and MVAs, a neuropsychological explanation could be advanced for the role of organic factors that might produce the mood and panic disorder symptoms. Both perspectives contribute to the process of understanding a complex symptom presentation.

Although populations, types of traumas, and comorbidity have thus far received separate discussion, the previous hypothetical case description illustrates how these factors may commonly combine with each other. In practice, clinicians and researchers assess and treat the aggregate effects of these multiple comorbidities, thus reducing somewhat the utility of addressing them in isolation. Instead of disentangling the effects, considering them as bundled for both research and clinical purposes may be more prudent. As more research focuses on the shared symptoms and etiologies of trauma. related and neurological conditions, descriptive models will evolve into more comprehensive formulations, including brain-function bases for symptoms of overlapping disorders.

EXISTING LITERATURE ON THE NEUROPSYCHOLOGICAL EVALUATION OF PTSD

The published studies examining neuropsychological test performances from PTSD patients to date are few and vary in format from sin-

gle clinical case to small group designs. As a group, these studies utilize a range of PTSD samples and administer both standard and nonstandard test batteries. Although existing studies have reported variability in cognitive problems among trauma samples, clinical observations suggest that PTSD symptoms show the most overlap with the neurocognitive domains of attention, memory, and executive functioning. Tests assessing some or all of these domains are included in the studies reviewed here. Selected relevant studies have been divided into combat trauma and noncombat trauma groups and reviewed briefly. A general discussion of points common to the group of studies follows at the end of this section.

Studies with Combat Veteran Populations

Gil, Calev, Greenberg, Kugelmass, and Lerer (1990) noted that empirical data on memory and cognition in PTSD were lacking, but reports from clinical studies document deficits in these domains across PTSD groups and across time (Archibald & Tuddenham, 1965; Burstein, 1984, 1985; Bleich, Seigel, Garb, & Lerer, 1986). In a review of studies employing the Luria–Nebraska Neuropsychological Battery (LNNB) to assess psychiatric patients, Moses and Maruish (1988) reported on results from a group of 36 POW survivors of the Bataan Death March during World War II. Findings from the LNNB were in the normal performance range for their age and education levels. Although several of the subjects were noted to exhibit a variant of PTSD, no diagnostic information was given, and the extent of PTSD in the group is unknown.

Gurvits and coinvestigators (1993) studied 27 medication-free PTSD Vietnam veterans and 15 non-PTSD combat controls via neuropsychological testing, neurological examination, and sleep-deprived EEG methods. All subjects were without substance abuse or dependence during the previous year. Although the PTSD group showed more neurological soft signs (NSS) than non-PTSD subjects, neuropsychological testing revealed few differences, and obtained differences between groups did not fall into the impaired range. Nine of the neuropsychological tests were significantly correlated (using Bonferroni-corrected *p*-values) with presence of NSS.

Uddo et al. (1993) compared 16 outpatient male, PTSD veterans with 15 National Guard enlistees on a battery of verbal memory, visual memory, and attention/concentration measures, including the Auditory Verbal Learning Test, Rey–Osterrieth Complex Figure Test (ROCF), Oral Word Association Test (Verbal Fluency), and the Digit Span and Visual Span subtests from the Wechsler Memory Scale—Revised. The PTSD group scored significantly lower than controls on measures of new learning, immediate recall, and delayed recall in verbal and visual modalities. No differences were found for Digit Span and Verbal Fluency. The pattern of performance characteristics was noted to be similar to patients with identified fronto-subcortical pathology. The findings could not he attributed to comorhid neurological disorders, because these conditions were excluded from the study.

Sutker, Allain, and Johnson (1993) employed twin-study methodology to investigate the effects of differential exposure to war trauma on psychological complaints and cognitive functioning in a twin pair of Army pilots from World War II, one of whom was shot down and captured as a POW. Measures of cognitive functioning included the Wechsler Memory Scale—Revised, the Wechsler Adult Intelligence Scale—Revised (WAIS-R), Categories Test, Wisconsin Card sorting Test, Porteus Maze Test, Trailmaking Test, and the modified Rey Auditory Verbal Learning Test. The POW twin was described as meeting lifetime PTSD criteria, but not current PTSD; the control twin had never experienced PTSD symptoms. The test performances of the POW twin were characterized as showing deficits compared to his brother and to education-matched norms on arithmetic calculations, visuospatial analysis, organization and manipulation, memory for visual material, planning and inhibition of impulsivity; and complex concept formation, conceptual shifting and cognitive flexibility. Considered within the context of his WAIS-R Full-scale IQ of 118, the POW twin's scores were relatively lower than what would have been expected given demographic factors and his brother's scores. The findings were hypothesized to reflect an acquired dysfunction involving the frontal lobes.

McNally and Shin (1995) examined 105 Vietnam combat veterans using the Shipley Institute of Living Scale (SILS) to compute WAIS-R Full Scale IQ estimates. Multiple regression analyses showed that IQ predicted 3% more of the variance in PTSD symptoms beyond variance attributable to combat exposure and years of education. The authors also reported that the lower a subject's intelligence quotient, the more severe the PTSD symptoms. Although the regression analyses were statistically significant, the actual benefit may not manifest clinically considering the multifactorial nature of IQ. The SILS is a completely verbal measure from which an IQ estimate that has a reasonably good correlation with WAIS-R Full-Scale IQ scores can he derived. In PTSD samples with learning disabilities that produce significant splits between Verbal IQ and Performance IQ scores, the SILS-IQ estimate will likely correlate less well. The protective influences of IQ in PTSD populations needs further elaboration.

Dalton, Pederson, and Ryan (1987) conducted neuropsychological testing on 100 male Vietnam veteran inpatients being treated in a specialized PTSD unit. All veterans received the Information, Arithmetic, Similarities, Picture Completion, Picture Arrangement, and Block Design subtests of the WAIS-R; 51 received the remaining subtest for a complete WAIS-R administration. Additional tests in the battery included the Rey Auditory Verbal Learning Test, Temporal Orientation, Serial Digit Learning, Trailmaking Test—Parts A and B, the Stroop Color–Word Naming Test, and the SILS. Few deficit performances were detected in this sample, with most mean test values being comparable to scores from normal, nonclinical samples. Only Trails B, Benton Visual Retention Test, and the Stroop Color–Word Naming Test showed slightly reduced group performances, and these findings

were interpreted as reflecting mild anxiety effects, as would be expected in psychiatric inpatient samples.

Gil et al. (1990) evaluated the neuropsychological test performances of 12 PTSD, 12 matched psychiatric controls, and 12 normal controls. The PTSD sample was drawn from the Jerusalem Mental Health Center outpatient clinic and had traumatic histories involving battles, terrorists attacks, and car accidents. The test battery consisted of the WAIS, the Bender–Gestalt test, Benton Visual Retention Test, the Mental Control and Paired Associate items from the Wechsler Memory Scale, a Hebrew version of the verbal fluency test, the ROCF, and the Continuous Performance Test. PTSD patients showed significant impairment relative to controls, but at similar levels of impairment to the matched psychiatric controls on all tests. The pattern of findings reflected a meaningful decrease compared to premorbid abilities, and the decrease could not be attributed to acquired neurological injury or substance abuse because these conditions were excluded from the study. The authors interpreted the findings as supporting the hypothesis of a general cognitive dysfunction, rather than a PTSD-specific dysfunction.

Everly and Horton (1989) reported pilot data on 14 patients administered one test—the Four-Word Short-Term Memory Test, which is a modification of the Peterson Paradigm. No sample demographics were provided, and no information on type of trauma was given. Potential age effects were disconfirmed by an age-split analysis of the scores. Results indicated that 12 of the 14 subjects showed short-term memory deficits.

Studies with Noncombat Trauma Populations

Literature on trauma in noncombat populations mostly includes experiencing or witnessing accidents, natural disasters, interpersonal violence, and the various forms of child abuse. Tarter, Hegedus, Winsten, and Alterman (1984) examined 101 delinquent adolescents referred by juvenile court for neuropsychiatric assessment, which involved structured cognitive testing, and compared the physically abused delinquents to nonabused delinquents. The abused delinquents had significantly greater histories of paternal and maternal alcoholism, parental criminality, physical abuse, and temporary foster home placement. Although no differences were found due to gender and race, the abused delinquents obtained statistically lower scores on an auditory attention span test for words, Trails A, Purdue Pegboard—nondominant hand, PIAT Reading Comprehension, WISC-R/WAIS Full-Scale IQ, Verbal IQ, three Verbal subtests—Vocabulary, Comprehension, Similarities, and the Arithmetic subtest. These findings reflected primary difficulties in verbal or linguistic processing realms.

Dinklage and Grodzinsky (1993) investigated the hypothesis that excessive, repetitive trauma can produce alterations in synaptic sensitivity, eventually producing depression of synaptic habituation and discrimination,

neuronal changes, and possibly neuronal death. To examine this hypothesis, 10 chronically traumatized children ages 8–15 were compared with 10 gender-, age, and IQ-matched controls on neuropsychological tests, including WISC-R, Continuous Performance Test, Story Memory, Sentence Repetition, and the ROCF. Results showed that relative to the matched psychiatric controls, the group of abused children had higher levels of inattentiveness, poorer impulse control, and below-average verbal memory. These finding were not apparent from the standard IQ measures. Of particular note was the fragmented production style demonstrated on the ROCF, which is a complex stimulus. The piecemeal approach suggests difficulty in either perceiving the larger basic structure separately from the details, imposing an organizing structure when reproducing the whole figure, or both. A similar limitation working with visuospatial stimulus configurations has been observed for adult borderline personality disorder patients.

The effects of severe abuse during early childhood on personality formation and brain development are unknown, but of increasing interest to researchers who observe correlations with *PTSD* (Famularo, Kinscherff, & Fenton, 1991; Kiser, Heston, Millsap, & Pruitt, 1991; Rowan & Foy, 1993), *borderline personality disorder (BPD), multiple personality disorder (MPD),* and *potential neuropsychological deficits* (Andrulonis, Gluck, Stroebel, & Vogel, 1982; Glod, 1993; Knoll, 1993; O'Leary, Browers, Gardner, & Cowdry, 1991; O'Leary and Cowdry, 1994; Silk, Lee, Hill, & Lohr, 1995; Teicher, Ito, Glod, Schiffer, & Gelbard, 1994). Swirsky-Sacchetti and colleagues (1993) compared neuropsychological test performances of 10 female BPD patients to 10 age- and education-matched nonpatient controls. The BPD group obtained lower WAIS-R Full-Scale, Verbal, and Performance IQ scores, plus demonstrated impairments in motor skills, figural memory, complex visuomotor integration, and freedom from distractibility. The results were interpreted as reflecting dysfunction in fronto-temporal regions.

O'Leary and Cowdry (1994) reviewed findings across four studies presenting neuropsychological test data on BPD subjects and concluded that as a population, no obvious impairments in verbal skills or reasoning were present. However, when performances were compared with matched healthy controls, the visuospatial skills and memory of BPD subjects were impaired relative to their education levels, IQ, and general neuropsychological functioning. The visuospatial deficits were interpreted as revealing problems with inhibiting attention to irrelevant information and selecting visual details from a complex configuration. Memory deficits were not sufficiently explained by immediate versus delayed recall, or deliberate versus incidental memory dimensions. Deficits also were not confined to emotion-laden material and suggested broader memory problems. No localizing pattern emerged, although fronto-temporal involvement was suggested by the findings. Etiological factors for possible underlying organic determinants were not posited.

Rosenstein (1994) reviewed the literature on neurophysiological corre-

lates in MPD and presented corresponding neuropsychological data from two clinical cases. Test findings for the MPD patients were interpreted as showing above-average overall intelligence scores with significant WAIS-R Verbal–Performance IQ differentials (> 23 points), and impaired free recall on memory tests. Severe depression and mildly abnormal EEGs were also present. Stringer and Cooley (1994) presented cognitive testing data from a case study of an MPD female with 2 female and 6 male alternate personalities. Two of the "adult personalities" each received a separate administration of the 20 subtests of Comprehensive Ability Battery (CAB) on different days. In addition, experimental attention measures, including visual digit span and the digit–consonant divided attention task, were administered to the personalities under separate (only one of the two personalities present), and "copresent" conditions (both personalities reported as simultaneously present in consciousness). Thus, each of the two "personalities" was administered three trials. The three trials consisted of one in which visual digits were monitored, one in which auditory consonants were monitored, and one in which both stimulus sources were monitored simultaneously. The expected decrement in tracking accuracy under the dual-stimulation condition is assessed against the baseline for single-stimulus presentation trials. For the "copresent" condition, in which dual stimuli were presented simultaneously, one personality was instructed to monitor presentation of the visual digits and the other personality instructed to monitor auditory consonants. The results showed individual variation across the 20 subtests of the CAB for the two personalities, but they were generally in the average-to-superior range on most tests except spatial ability, perceptual speed, and accuracy, which were low-average to below-average. Findings for the divided-attention task when only one personality was present showed the expected performances decrement for the dual-stimulus presentation condition. However, performances under the "copresent" condition showed no decrement in tracking performances compared against baseline, single-task performances. Assuming that two personalities present accounted for the performance, the findings suggest the possibility of a cognitive superiority for divided attention when the copresent personalities each monitored one component of the simultaneously presented dual stimulus, much like reciprocally aware, parallel processors consciously attending to the environment.

Summary Discussion of Studies

Overall, the findings in these studies are inconsistent regarding the presence and extent of neuropsychological impairments in PTSD and trauma-related disorders. Symptom complaints from PTSD patients usually include attention/concentration, memory, and some executive dysfunction. Although various studies detected these problems, as a group the studies reviewed did not consistently find impairments in these domains. However, drawing general conclusions from across the group of studies is problematic, because many

of studies reviewed here are descriptive or uncontrolled case studies. The impact of the studies as a group is affected by methodological and sampling issues, the limits of the case-study format, and problems with the group designs. In general, the reporting of basic information was variable. Specifics about PTSD diagnostic procedures, trauma-history evaluation methods, and testing protocols ranged from missing or sparse to incomplete. Studies often noted that patients or subjects had PTSD without reporting specific PTSD symptoms or other general trauma-history variables to describe the subjects, such as presence of multiple traumas; chronicity of symptoms; or severity, as defined by frequency, duration, and magnitude of the stressors. Findings were often not interpreted relative to a conceptual model. The clinical case studies typified uncontrolled investigations suffering from the typical threats to internal and external validity (Campbell & Stanley, 1963). Their main contribution is the demonstration of neuropsychological assessment of comorbid conditions and PTSD that suggest further issues to explore.

Conclusions derived from the factorial designs are variously constrained due to design and methodological limitations, including (I) small sample sizes; (2) poorly specified inclusion and exclusion criteria, if present at all; **(3)** inadequate matching of the control group on relevant variables such as gender, age, education, type, and severity of psychiatric disorder; substance abuse parameters — drug type, pattern of drug use, duration and severity of use, time since start of sobriety (if sober); and (4) nonequivalence on cohort factors such as service era and military experience, medical health status, and comorbid neurological status. Some studies reduced the need to match on these variables by employing a priori exclusion criteria.

Generalization across group studies is limited due to discrepancies in findings possibly related to nonequivalence of samples for the aforementioned reasons, and parameters of the traumatizing event(s), which are not addressed sufficiently in standard neuropsychological protocols. To increase rigor in the group designs, matching must occur for the relevant variables, and these variables will be both trauma- and neuropsychologically based when studying cognitive problems of PTSD. Group studies that blend the variants together, disregarding appropriate matching factors, may obscure the differences within their sample that truly represent trauma effects on cognitive abilities.

None of the studies reviewed reported methods to account for potential reactivity to the testing process, or whether reactivity was measured in their studies. For some of the studies reviewed, it is possible that findings reflect altered engagement with the tasks during the data collection (cf. section on reactivity to the testing process for examples). Manifestations of reactivity to the testing process may vary widely across diverse groups, such as accident and crime survivors, combat veterans, and MPD patients. In studies where deficits in attention and memory were detected, it is unclear whether *a* true deficit was found or whether competition for attentional resources due to reactivity interfered with memory. Determining the difference is im-

portant for addressing the issue of stability of the impairments detected. On short-interval retesting, deficits caused by reactivity should reverse unless identical reactivity is elicited again.

In the research studies with identified, comorhid neurological conditions, the designs limited the ability to demonstrate an incremental effect for PTSD above the effects known to result from the neurological condition alone. Test batteries varied in composition, and the absence of standardization restricts comparisons across studies. Some greater degree of standardization needs to be developed without impeding the discovery of the unique contributions of trauma effects to the nenropsychological test results. Clinical protocols striving only to determine if cognitive processing problems exist may be secondarily concerned with incremental validity, hut this is of greater concern for defining the components of a model.

The studies reviewed represent initial attempts to understand the functional impact of PTSD on brain–behavior correlates. Although the collective findings present a mixed picture at this point in time regarding consensus effects of trauma, they highlight the need for conceptual and methodological refinements that will shape future studies. Addressing these aspects will produce parallel benefits for practical issues surrounding test interpretation.

TEST INTERPRETATION ISSUES

Previous discussion related to types of comorhidity and neurobiological substrates is germane to the interpretation of test data, because these factors form a significant part of the context for determining presence and absence of deficits, for analyzing variations in test performances, and for developing conclusions and recommendations. The bulk of the interpretive context is derived from the history provided by the patient, collateral interviews, and the medical record. Obtaining a good neuropsychological history cannot be stressed enough. Although emphasizing solid history taking is not novel, suggesting that neuropsychologists include trauma histories and traumatologists include neurological factors to increase comprehensiveness may be a new perspective. For neuropsychologists who administer a standard battery each time, the additional history will primarily assist during the interpretation process. For those who employ a flexible battery approach, the comorbidity information may influence initial test selection as the test battery is composed.

Preinterpretation Considerations

Some general points, commonly understood to he important for neuropsychological test interpretation, have increased significance when testing PTSD patients in clinical settings.

1. ***Recent sleep quality and sleep patterns should be assessed.*** Chronic sleep problems are often present in PTSD patients (Woodward, 1993), including sleep apnea (Boza, Trujillo, Millares, & Liggett, 1984) and REM sleep behavior disorder (Lapierre & Montplaisir, 1992). Neuropsychological deficits resulting from hypoxia secondary to sleep apnea range from global, diffuse cognitive dysfunction to isolated memory problems (Greenberg, Watson, & Deptual, 1987; Martzke & Steenhuis, 1993). Other sleep problems are due to increased sleep onset latencies and midsleep awakenings from regularly occurring nightmares. Alcohol and drugs may be taken in extreme amounts to promote sleep. In some, the emotional aftereffects of the trauma-related nightmare persist into the next day and may result in sustained increases in hyperarousal, hypervigilance, and flashbacks. Daytime naps may be substituted to compensate for lost nighttime sleep, thus affecting circadian rhythms and potentially the fatigability during the test battery.

2. ***Residual effects of peripheral physical damage as a result of the traumatic experience should be systematically reviewed*** (e.g., motor vehicle accidents; torture survivors—ears, eyes, fingers; and military veterans—damage from explosions, booby traps).

3. ***Medication and psychoactive substance use should be assessed as well for pattern of usage and any recent change (increases or decreases).*** Fluctuations in usage patterns may parallel phasic changes in PTSD symptoms or be related to anticipatory anxiety about the testing process. For some, the increased medication or psychoactive substance use may facilitate test performances by reducing anxiety, whereas in others, excessive use detracts from test performances.

4. ***The role of compensation-seeking, litigation, and secondary gain needs to be considered.*** PTSD is a compensable disorder in the VA system, in workers' compensation claims, and in lawsuits. Recent controversies over PTSD in the courtroom and false memory syndrome in childhood sexual abuse cases could focus added attention on nontest aspects of the neuropsychological assessment process in forensic cases. In VA settings, receiving compensation is associated with service and monetary support. Veterans are often vigilant for any possible evaluations suggesting a positive change in their disability status that might become part of their official charts and jeopardize their compensation rating. Including neuropsychological measures of malingering in the test battery will assist in evaluating suspected, intentionally manipulated performances.

5. ***Particular emphasis needs to be applied to evaluating attentional problems.*** Systematic investigation of attentional functions is generally required to establish the patient's ability to engage the tests. Intact attentional abilities are the foundation for higher cognitive information-processing abilities, including memory and language (Mirsky, Fantie, & Tatman, 1995). As attention is not a unitary construct, specific measures of multiple modes should be included in test batteries. This recommendation is of particular importance with PTSD patients who experience hypervigilance, intrusive recollections,

hyperarousal, and dissociation that have a high potential for disrupting engagement with the testing process. These considerations and the discussion points in the section describing reactivity to the testing process should be used to answer the following questions: Was the patient distraction-free when instructions were given, so that all task requirements, not just the gist of the instructions, were fully understood before the test began? Were all facets of attention adequately engaged during testing? Was adequate attention applied to each test?

In addition to the standard data analysis and test interpretation process, the neuropsychological evaluation should ultimately address these fundamental questions. First, do ***any deficits exist?*** This can be addressed by comparing the test performances to age- and education-matched normative values when available.

Second, ***when deficits are present, what*** is ***the pattern?*** The pattern of deficits can first be described as lateralized, localized, or diffuse relative to normal population values. Qualitative performance features can be characterized in a similar manner, and when available, evaluated against normative values. Once performance levels for each test are established, levels within and across tests can be compared for patterns of relative strengths and weaknesses in the neurocognitive domains sampled.

Third, is ***the pattern of deficits similar to other disorders, and is this pattern typical of PTSD populations?*** Obtained patterns of deficits can be analyzed against other known disorders; but given the high incidence of observed comorbidity, this exercise may have less utility in chronic PTSD samples. Unless a suitable set of comorbid comparison groups is available, determining whether the pattern is typical of PTSD will be more complicated. Based on absence of consistent findings from the review of literature in the previous section, deciding which variables or test patterns to use in the process is open to debate. Comparing findings across studies is difficult at present because of the lack of standardization of diagnostic methods; variability in sample demographics and characteristics, in testing environments, and in the batteries of tests administered. An insufficient amount of study has been conducted that controls for these relevant variables across various types of traumas and populations. Individual settings need to establish local normative values based on comparisons of PTSD versus relevant non-PTSD control samples of clinical interest. This approach creates comparison groups of regionally similar, neurologically normal samples, in addition to the typical neurological, psychiatric, medical, and non-PTSD samples.

Fourth, ***was the clinical testing process free from the effects of unintended affective priming?*** If present, did the priming produce a main effect across tests, or interact with various components of the test battery to produce differential responding? Although relevant clinical considerations for neuropsychological testing are presented in the section reactivity to the testing process, previous investigation by Zimering, Caddell, Fairhank, and Keane (1993)

demonstrated the effects of affective priming on task performance using an experimental paradigm. Exposure to auditory combat sounds produced subsequent decrements in performance on a motor steadiness task and letter vigilance during a continuous performance task. Corresponding increases in frightening and violent intrusive thoughts were also reported during the postprime intervals.

Fifth, ***when present,*** do *cognitive problems at the time of testing represent* ***an exacerbation of existing symptoms typical of the phasic variation of PTSD, or a stable level ofsymptoms for the PTSD patient?*** Do the observed cognitive problems covary with general level of distress and symptom severity; or, is there an interaction pattern among subsets of symptoms, with some deficits remaining relatively stable and others showing variation that is correlated with PTSD reexperiencing symptoms? Answering this question is a clinical consideration based on the history of symptom fluctuations. Symptom pattern variations serve as the context for integrating test findings during the formulation of clinical recommendations. For example, PTSD veterans can experience cyclical variation in symptoms related to seasons of the year, anniversary reactions, and publicly celebrated holidays. Variations in medical and psychological symptoms may prompt compensatory changes in medication use and substance abuse to modulate effects. The effects of an alcohol-abuse lifestyle can lead to traumatic brain injuries (TBIs) of varying severity from fights or motor vehicle accidents. TBIs are known to produce short- and long term increases in depression and anxiety, confusion, social withdrawal, as well as cognitive function changes. Thus, the level of test performances for any given administration could represent cyclical variation in symptoms that may regress to the mean on retesting, or alternatively, test performances that are influenced by recent, minor TBI. Neither may be reported spontaneously by the patient.

PTSD Status and Reactivity to the Testing Process

One of the underlying assumptions for validity of neuropsychological test interpretations is that maximal performances were obtained during data collection so that decrements in performance levels relative to comparison norms would be associated with brain-related conditions. Methods for determining the role of confounding factors that attenuate performance must be systematically employed by the evaluator. Standardizing the test administration procedures, creating a conducive testing environment, and minimizing error due to nontest related factors are some methods commonly incorporated for optimizing validity of the data collected. Psychiatric disorders, and PTSD in particular, require that additional considerations be addressed. For neurological disorders and syndromes in which the lesion is static or progressive, variations in the pattern of test performances are assumed to be attributable to the underlying lesion. In psychiatric samples with distorted reality testing, skewed information-processing tendencies, and

disturbances of mood, neuropsychological test performances can more proportionately reflect psychiatric status. With PTSD, where reactivity to environmental cues characterizes the condition, the neuropsychological testing process can interact with the patient's trauma history, resulting in performance levels that inaccurately represent true cognitive functioning. The literature to date has not adequately addressed the range of reactivity to the testing process that can exist when assessing PTSD patients.

Interaction of Trauma Histories and Administrator Characteristics

Where PTSD Criterion A traumatic events occurred within an interpersonal context such as physical violence, rape, or threat to life by another person, patients will report both general and specific characteristics of the perpetrator or assailant. These details and general descriptions constitute cues that function to evoke physiological arousal and memories of the trauma when encountered in the everyday environment. The interpersonal context for testing is often a small office where the patient meets with the test administrator, who by virtue of gender or race alone may he a triggering stimulus for recall of traumatic experiences (e.g., Asians—for Vietnam veterans, or males—for sexual assault survivors).

Beyond general characteristics, trauma survivors may remember small details about the perpetrator that have become associated with threat or may have served as warning signals cuing escape/avoidance in the past. For instance, incest survivors who were abused when the perpetrator was intoxicated mav have learned the connection between the bloodshot eves associated with drinking and an increased probability of molestation. Should a test examiner's eyes resemble the perpetrator's (i.e., bloodshot, possibly due to allergies or wearing contact lenses), intrusive memories or dissociative episodes of varying lengths may be unintentionally triggered. Smells and odors can also function as powerful memory retrieval cues for trauma survivors. What would, under usual circumstances, be benign characteristics of the examiner may become significant in the testing process. This scenario could apply to men and women who might have been incest victims during childhood and to military veterans who experienced sexual assaults in the service. Although sexual assault is more commonly considered an act perpetrated by men toward women and girls, reports of mother–son, older females–younger boys, and men assaulting men in the military are reported in clinical populations. Without knowledge of the history of sexual assault, the stimulus value of the test administrator and the examinee's subsequent reactivity during testing may be undetected or, if detected, misattributed. Disengagement with the testing process by the examinee may be as subtle as a focused stare at the test stimuli and increased latencies to respond during the task to a full dissociation or panic attack.

Interaction of Trauma Histories and Testing Environment

In other instances, a number of features in the testing environment combine to produce a disruption in the examinee's level of task engagement. For example, if a trauma such as being a prisoner of war, an elevator accident, a car accident, or sexual assault involved being in a confined space, the combination of a small testing room and features of the test administrator may elicit a range of reactions, including intrusive memories, increased physiological reactivity, and dissociation. Adequacy of the neuropsychological testing environment on a number of levels is always an issue needing attention. Room space for clinical activities in most settings is limited, and assessments are often conducted in rooms not specifically designed for testing. The features of the room space can be very important for PTSD patients. If the room is not sound-isolated, noise in the hallway or adjoining rooms may repeatedly draw the attention of the hypervigilant patient. Spurious noise problems are frequently addressed by masking them with white-noise generators or small fan devices in the room. Although successful in blocking other noises, the constant low-level din emitted from the masking device can resemble the characteristics of background noise from past traumas and may trigger memories for patients who were in chaotic combat, accidents, or natural disasters. Combat veterans exposed to the concussive effects of artillery explosions and torture survivors may have chronic tinnitus that is compounded by the masking devices (Graessner, 1993).

Chronic PTSD patients who have developed a pattern of attending to stimuli around them and then escaping cognitively from triggered intrusive images may automatically engage in these monitoring processes and escape strategies during testing without reporting them. Depending on the test being administered at the moment, the effects on performance could include slower reaction times, longer times to completion, interference with storage during memory tasks, incomplete processing of instructions for the task, the appearance of inconsistent responding, or premature discontinuation of the task by the patient, which might look like "giving up." Examiners should be aware of these instances, but, unless reported freely by the patient or when prompted, they may go undetected.

Another aspect of the testingenvironment that might interact with trauma histories is silence. For a portion of trauma survivors, a conditioned emotional response may be elicited by silence. For these individuals, silence or hiding in silence may have followed or preceded the onset of a traumatic experience. Combat veterans who waited in silence while setting an ambush, or ones who noticed the silence before being ambushed on patrol, may become more agitated by quiet in sound-reduced testing rooms. Incest and crime victims who hid in silence may have similar reactions. Silence is also an environmental condition that offers nothing for the PTSD patient to monitor, and without a stimulus present to capture attention, unwanted thoughts of the trauma may intrude. To prevent silences, patients may talk at fairly

continuous rates and provide overdetailed answers to free-response sections of tests.

Interaction of Trauma Histories with Test Features and Task Requirements

Reactivity in this category is not necessarily intuitive if the test behavior of neurological patients is the primary comparison sample. Patients with dementias, cerebral vascular accidents, and neoplasms will infrequently have direct traumaticassociations with features of the test stimuli or the task. Frustration and some catastrophic reactions can be observed when these patients notice poor performances, hut idiosyncratic associations are more rare. In PTSD patients, however, a generalization gradient may have developed from a specific trauma-event cue or set of cues to broader classes of stimuli. The neuropsychological test examiner may encounter unexpected reactions to the test materials that are associated with traumatic experiences. **A** case example of a military combat veteran illustrates how test characteristics interact with patient history.

During administration of the test battery, a veteran whose trauma involved a near-death experience from artillery fire had a series of responses to the neuropsychological tests that culminated in discontinuation of the testing because of the level of distraction he experienced. During a computerized administration of a Continuous Performance Task—which requires sustained monitoring for, and response to, the appearance of an X in one condition, or an X which was preceded by an **A** in another condition—he developed heightened levels of physiological arousal because the colored, letter stimuli of the task in his view "exploded" onto the screen. His vigilance became heightened, and memories of the shelling he experienced were evoked. Once primed by these memories, subsequent testing was affected. During the Visual Span task from the Wechsler Memory Scale—Revised, he again experienced an intrusive recall of his traumatic experience. This test, which is a visual analog of the Digit Span task, required the patient to observe and reproduce a sequence that is finger tapped by the administrator on small, colored squares printed on a stimulus card. The Forward Span test uses a card with green squares and the Backward Span task uses one with red squares. The process of reproducing the tapped sequence on the red squares reminded him of red tracers from small arms and artillery fire. During motor speed testing for the Finger Tapping test, his association was to past experiences of squeezing the trigger to fire a machine gun while on guard duty. During this association, he raised his head, looked out the window and had a momentary flashback of the landscape around his guard post. The effects of these experiences lingered and manifested during the Sensory–Perceptual Examination, producing sensory errors that were inconsistent ipsilaterally and contralaterally. Subsequent retesting of sensory–perceptual functions on another day produced a within-normal-limits performance. Had

he not provided the information on the distracting intrusions he experienced, the inconsistency in his performance could possibly have been misconstrued as motivational problems or malingering.

Computerized tasks, such as the Continuous Performance Test, that present stimuli at short, repeating intervals may elicit a dissociation in patients who are prone to this experience. Explanations for this are unclear, but this response during testing is an acute, reactive state that limits the potential of patients to generate their best performance. It may be that if targets are presented at a rate that is too fast, the patient may feel unable to process the information, producing feelings of failure, inadequacy, and helplessness, which may be associated with past trauma. Alternatively, the dissociative experience may be a manifestation of a photosensitive seizure or a reflex epilepsy. Photosensitive seizures can result from the presentation of synchronized visual stimuli such as a flickering light in specific frequency ranges. Monochromatic red light has been shown to be more potent than other colors (Engel, 1989). Complex reflex epilepsies are elicited by very specific stimuli or stimulus conditions that frequently involve some level of cognitive or emotional appreciation of the stimulus (Forster, 1977).

The previous discussion illustrated how the visually oriented test stimuli can elicit reactions; not being able to see can be equally distressing. Neuropsychological procedures that require wearing a blindfold (e.g., Tactual Performance Test) may be problematic for survivors of nighttime physical or sexual assault, or for veterans with combat experiences such as being ambushed in the dark, being assigned to explore enemy tunnels in Vietnam, being blindfolded and tortured as prisoners, or having temporary loss of sight from explosions or concussions. More generally, wearing a blindfold increases feelings of vulnerability by reducing patients' ability to be vigilant of the environment, and these feelings in turn may exacerbate PTSD symptoms during testing.

Thematic Associations Cued by Testing

The testing process may activate a variety of thematic associations with traumatic experiences in PTSD patients. Conceptual themes involving the absence of prediction and control may be aroused during assessment and manifest as overt discomfort and agitation when faced with ambiguity. It is not uncommon for PTSD veterans to report anticipatory anxiety beginning days before an appointment in which a new procedure or an anticipated negative experience is expected. Their distress related to these themes can often be traced to past anticipation of engaging the enemy and the need for information to maximize prediction and increase the likelihood of survival. Anticipation can prompt a search for information that may increase distractibility and confound performances on the initial tests in the neuropsychological battery. Alternatively, a proportion of veteran samples also report that they become less anxious and very focused once the objectives

of a task or situation are clear, similar to battle conditions in which their attention became acutely focused once the fighting had begun. These individuals may show greater freedom from distractibility on portions of the testing.

Themes related to survivor guilt, acts of commission and omission (atrocities committed or witnessed and not prevented), trauma contexts, and low self-worth may trigger further associations with more specific memories. Poor test performance can activate cognitive schemas surrounding feelings of guilt associated with responsibility for the consequences of underperforming in the past (e.g., negative consequences to others, or when lives were at stake). If patients notice that their performance is flagging, followed by feelings of "failure" that cue recall, then trauma memories may intrude during task execution. Low self-esteem and feelings of low self-worth may interact with the intrusive memory to further depress or interrupt test performances. As an example, after fairly easily completing the Vocabulary section of the SILS, then experiencing much difficulty on the Abstract Reasoning section, a combat veteran was overcome by visual and verbal intrusions of leading his men in a failed mission. Feeling failure on the test cued the intrusions during the SILS; some of the intrusions were auditory, such as "Don't fail; don't let the men down."

Performance anxiety can also induce physiological arousal, and these states can subsequently trigger intrusions during testing. During traumatic experiences, physiological reactions can be a very prominent aspect of the experience (e.g., feeling or hearing their heartbeat in their ears may have been paired with the silence of sitting still and keeping quiet during an ambush in Vietnam, or with hiding from sexual abuse perpetrator). If an error-free performance, defined as either exhibiting or inhibiting an action, was linked to survival in the past, the similarity of task requirements during testing may activate this schema. This possible interaction should be considered for tasks in which exhibition and inhibition of specified responses are tested (e.g., motor Go/No-Go tests), and for procedures in which direct feedback in the form of correct–incorrect is standard during the task (e.g., the Wisconsin Card Sorting Test, the Categories Test).

Single words may be sufficient to cue a theme. Previous findings using the Stroop paradigm (Litz & Herman, 1993; McNally, Kaspi, Riemann, & Zeitlin, 1990) have shown increased latencies for color naming of trauma-relevant words in combat veterans. These findings have been interpreted as representing not only past exposure to trauma, but also the severity of intrusive symptoms during the task (McNally, 1995). Language assessment tasks may inadvertently trigger associations. For the Controlled Oral Word Association procedure, which measures verbal fluency by asking patients to produce as many words as possible starting with a target letter, traumatized patients may generate words directly related to their trauma that prime affect- and cueintrusive recollections (e.g., words beginning with "S" for combat veterans: shrapnel, shelling, sharpshooter, sniping).

Idiosyncratic responses to other tests have been observed in combat veterans. Items in Subtests IV, V, and VI of the Categories Test include configurations that can resemble defensive physical perimeters of military compounds as viewed from aerial maps. Establishing defensive perimeters and planning missions that targeted enemy positions often involved detection of pattern configurations. To some, Mesulum's Letter and Shape Cancellation task resembles a schematic of a minefield. The recall trials of the ROCF and the Visual Reproduction of the Wechsler Memory Scale can remind some of diagrammatic renderings of enemy compounds. Spacial associations, linked to visual memory, may rapidly induce intrusions.

Receiving instructions for completing neuropsychological tests has been reported by some veterans as feeling like being briefed for an upcoming mission. As they listen to the instructions from the examiner, their attentional focus narrows so that the objectives would be clear. In some this narrowing of focus leads to an intense engagement with the task, and they may overly associate the moment with past military experiences. In others, if closely associated with a trauma experience, it may subsequently be distracting. Using the computerized Continuous Performance Test (visual X/A–X version) again as an example, a veteran reported that listening to instructions elicited a "mission-mode" in him as he prepared to engage the task. Task instructions that required him to respond to target letters as quickly as possible on the screen paralleled mission requirements to quickly spot an enemy soldier in the bushes/jungle and shoot before being shot ("It was like shooting at something coming out of the dark"). Pressing the computer key on this task was analogous to pulling the trigger. Assuming a *mission mentality* produces a narrowed focus of attention that either facilitates or impairs test performances. Shorter latency commission errors can increase from too rapidly responding to *any* on-screen stimulus change (the "shoot-first" strategy); while omissions errors ("missed targets") can distressingly induce feelings of being vulnerable because of poor vigilance (symbolically shot by the enemy).

In hospital settings where PTSD patients with medical problems are being tested, feelings of vulnerability resulting from having a serious illness can induce increased reexperiencing and generalized arousal that may be present during testing. In the hospital context, patients may be shuffled through clinics and receive procedures with little explanation and little feedback about test results. If prediction and control are reduced, a decreased sense of rapport with the care providers can result in a distrust in the process. Feelings of vulnerability can be heightened in the absence of trust. For trauma survivors whose traumatic events involved violations of interpersonal trust, or those who have become highly distrustful subsequent to the trauma, the testing process may evoke emotional reactions. Abused children may distrust adults, and adult trauma survivors may distrust authority figures, government, institutions, hospitals, and staff affiliated with medical centers.

Impersonal treatment, which can be part of the patient experience in large institutions, often inflames existing agitations. Military veterans are known to express feeling of being experimented upon, and some have documentation of their military treatment (e.g., radiation exposure from being placed in open trenches during nuclear bomb testing). The word "testing," in neuropsychological testing can elicit reactions, because it seems like a direct challenge, or something that must be endured. General negative associations with testing may be present for many patients who had poor academic histories or aversive experiences taking tests in school, and may be particularly true of trauma survivors with learning disabilities. Strategic attention applied to developing good examiner–patient rapport is central for reducing potential arousal that accompanies feeling of vulnerability from these factors.

Whereas the types of themes discussed here may be shared to a degree by many patients as reactions to illness or as part of normal life, for traumatized patients they may be more readily activated because of their past connection with life-threatening events producing traumatic stress reactions. Chronic PTSD patients may not necessarily be aware of the relationship between past experiences, cue-evoked themes, and the impact on their neurocognitive abilities when themes are cued during interviews or testing. Although awareness of some impact may occur if physiological arousal or feelings of discomfort increase, the patient may causally attribute these reactions to the testing process rather than past trauma experiences.

Accounting for the Potential Process Confounds

The degree to which these parameters alter neuropsychological test results is not well researched, although general performance enhancement is an unlikely outcome. The process of conducting neuropsychological testing with PTSD populations for clinical or research purposes should include mechanisms for addressing the presence of altered states of engagement with the testing. The test administrator needs to be clinically aware of the general range of potential reactions from traumatized populations, and be informed of the specific types of reactions the individual patient might exhibit during the course of testing, based on an understanding of their trauma experiences. Testing may need to be tailored accordingly. Although some patients with PTSD have levels of hypervigilance requiring a clear view of exits—examiner's back to the door, others may need a physical arrangement of the furniture that allows them the perception of clear egress if necessary—patient's back to the side wall.

Levels of arousal need to be carefully observed as signs that attention may be shifting internally to intrusions or externally as an increase in hypervigilance. Preparing patients for testing by providing a thorough explanation of the upcoming test procedures facilitates them in predicting the experience and may reduce the anticipatory anxiety. Eliciting their partici-

pation in reporting attentional shifts to the examiner increases the likelihood of detecting shifts. Succeeding here may be a challenge that depends in good measure on rapport with the test administrator, assessment team, or primary referral source. One salient feature of PTSD patients is avoidance of stimuli and circumstances that arouse memories and physiological reactivity. Self-observation of, and comfort talking about, their symptoms may not be a well-developed part of their behavioral repertoire, especially if increased anxiety results from monitoring and disclosure. To ensure the validity of test interpretations, process checks should be included with data collection so that patterns of deficits can be correctly attributed to reactivity when present, rather than to underlying lesions.

Patients may not be able to tolerate a lengthy battery of tests. A common consideration for geriatric populations and others with fatigue-prone disorders, PTSD patients should be monitored for fatigue levels as well. Veteran populations have been described as "commonly reporting fatigue, concentration difficulties, somatic distress, and other complaints that impair capacity to endure lengthy testing sessions" (Dalton et al., 1986, 1989). Monitoring for fatigue is a good assessment practice in general. For the more chronic groups of PTSD patients with greater symptom severity, the cognitive demand of sustained, focused, mental activity may produce fatigue, accompanied by increased hypervigilance and vulnerability to intrusive recollections as their cognitive capacity to inhibit these reactions wanes.

To minimize fatigue for clinical evaluations, long batteries can be divided into shorter sessions. Although this assists in maintaining peak performance for each session, it complicates the correlation of test findings across sessions, because the patient is open to effects of environmental events that may exacerbate symptoms and alter mood states. In controlled settings, acute shifts in clinical presentation will primarily result from sleep disturbance, nightmares, flashbacks, increased intrusions, carryover effects of therapy groups, and interactions with visitors. For outpatients, similar factors may be influential, with the added effects of substance abuse and increased use of prescription medication to control symptom escalation.

Blind interpretation of testing data from PTSD protocols can be confounded, because transient alterations in attention from intrusions or dissociations are difficult to rule in or rule out by examining the pattern among test performances. Attentional measures within the neuropsychological test battery are often examined to make a judgment about examinees' ability to concentrate, and if they fall within expected ranges, a generalized assumption of adequate attention across the test battery may be inferred. However, the transient nature of these alternations in attention and their occurrence as a result of specific cuing during testing makes this practice potentially problematic for establishing the validity of the whole test battery. To facilitate the data interpretation by the neuropsychologist, clinical psychometrists, trainees, and neuropsychological research technicians need to have structured-process recording forms available for recording their observations

during testing, and they should also be trained in observing the many diverse reactions demonstrated by PTSD patients.

FUTURE DIRECTIONS

With each advance in behavioral neuroscience methods, the potential for discovering psychopathological etiologies improves. Correlative measurement of cortical functioning via structured neuropsychological testing, electrophysiological assessments, and noninvasive scanning techniques offers opportunities for understanding the cortical activity of PTSD patients as they process information. Functional neuroimaging methods also provide a comparative standard against which functional neuropsychological deficits can be indexed. These methods all complement information obtained from psychological evaluations, structural neuroanatomical scans, and neurochemical assays.

In recent years, psychiatric disorders have been increasingly conceptualized as manifestations of brain dysfunction. PTSD may constitute a unique opportunity to investigate the effects of the interaction of environment and brain functioning on development of psychopathology. Unlike most other disorders in the DSM, the Criterion A component of the PTSD diagnosis explicitly implicates environmental experiences as causal agents in the symptom development process. The human brain as repository of past experience, primary processor of present experience, and architect of planned future experience is the wellspring of PTSD symptoms. Although current technologies and methodologies may limit the depths to which precise causal mechanisms can be probed in research protocols, many potential empirical investigations can be initiated to continue advancing our understanding of the complexities of PTSD's neurocognitive concomitants.

Different research goals may need to be defined, based on the intended applications of findings from the neuropsychological studies. If the goal is to identify the unique contributions of a single traumatic experience in determining the cognitive impairments associated with PTSD symptoms, then a relatively pure—unconfounded, noncomorbid—sample of trauma survivors needs to be studied. This sample should contain patients who meet Criterion A, are beyond the acute stress disorder stage, have developed sufficient PTSD symptoms to meet diagnostic criteria but have not yet begun a pattern of negative coping behaviors that would confound interpretations of the data, and are free of preexisting neurological problems and prior trauma. Findings here would conceivably add to our understanding of the cognitive effects of trauma in isolation. However, findings and models developed subsequently from this method of study may not generalize to typical clinical samples that are characterized by complicated symptom presentations. Alternatively, if comorbidity is presumed to occur at rates high enough to

be considered inherently associated with PTSD, especially in the case of chronic PTSD, then generalizability of the developed models will be enhanced by studying samples that also contain the comorbid characteristics of interest. Experimental designs that account for confounds by blocking them as levels of the independent variable or multivariate statistical techniques will need to be employed in these studies to disentangle relative causal influences of the target variables.

Model Development

A neuropsychological model of PTSD is needed to guide research investigations and clinical process. Future research into the origins of the current PTSD symptom clusters will likely find that they reflect dynamic patterns of whole-brain activity. What remains unclear is the balance between cortical and subcortical contributions to observed or reported PTSD symptoms. Having a well-defined model that attributes symptoms to hypothesized brain regions or brain systems would assist in focusing investigations, developing measures, and refining the assessment process. Neuropsychological tests, generally regarded as more sensitive to cortical brain activity, should be indirectly affected by the subcortical activity that may underlie the form and expression of some clusters of PTSD symptoms. Nightmares and triggered symptoms such as exaggerated startle response and physiological reactivity to reminders of the trauma are likely to be primarily associated with subcortical origins compared to conscious, effortful avoidance of thoughts, feelings, and circumstances reminiscent of the trauma, sense of foreshortened future, and concentration difficulties. However, if existing neurobiological models of PTSD are accurate (Kolb, 1987, 1988, 1989; Pitman, 1989a, 1989b), then the effects of the postulated neural hypersensitization process from traumatic stress exposure should influence multiple brain systems and be detectable across many levels of functional testing.

The challenge for developing a neuropsychological model of PTSD is defining the assumptions upon which hypotheses can be formulated. The relationship of test responses to the phenomenon of interest is only as valid as the established correspondence to brain functioning. Current conceptualizations of brain-function correlates of PTSD produce more questions than answers. Neuropsychological approaches will be influenced by developments in molecular and neurobiology that contribute to answering questions about the mechanisms of trauma's impact on the brain, affecting cognitive functions and altering the manner in which information is subsequently processed. If mechanisms can be discovered, then cortical status can be included as one of the preexisting vulnerabilities that may determine whether PTSD develops after exposure to **a** potentially traumatizing event.

A comprehensive formulation of the neuropsychology of PTSD would need to include the following to be viable as a heuristic for guiding research into the next century:

1. The model needs to address possible differences in neuropsychological test performances as a function of basic demographic characteristics such as age, gender, and developmental level (in children).

2 The interactions of demographic factors with the type and severity of trauma experienced may create unique subtypes that need to be predicted. Trauma researchers are currently struggling with the challenge of how best to specify the psychological effects for the complex combinations of frequency, intensity, and duration of trauma exposure. Adopting the most effective procedures for classification of trauma experiences will be important for constructing representative groups against which neuropsychological test findings can be indexed.

3. While current clinical research strives to define the distributed effects of trauma on psychological functioning for adults and children, the same process must be extended to neuropsychological functioning in PTSD. It is conceivable that early traumatic experiences affect the overall organization of the brain, producing deficits in processing some types of information, and possibly a corresponding superiority for other types of information. A very extreme example of this organization can be seen for idiot savants, whose severe limitations in overall intellectual development are matched by an equally extreme untrained, native ability in some circumscribed activity such as mental calculations, or playing a musical instrument. Stringer and Cooley's (1994) demonstration of superior divided-attention performances for copresent multiple personalities may be one example of distributed processing abilities resulting from a trauma-influenced cortical organization. If this type of organizational process occurs along multiple continua, then many potential versions of PTSD-related cognitive patterns might emerge on testing.

4. Clinical samples have repeatedly shown that PTSD symptoms do not exist in isolation; as such, the high rates of comorbidity must be explained, not Just viewed as a nuisance variable. **A** salient issue in this regard is the contribution of preexisting factors and posttrauma adaptation to the PTSD symptom presentation. For example, preexisting factors may promote acute stress responses, whereas posttrauma adaptations can produce chronic, maladaptive coping patterns manifesting as substance abuse as a method of managing the hyperarousal and reexperiencing. Having a current substance abuse lifestyle alone can produce cognitive changes independent of any hypothesized effects from trauma (Ardila et al., 1990; Mittenberg & Motta, 1993; Parsons, 1987), which highlights the point of studying symptom aggregates. Moreover, the various combinations of factors closely represent the actual patients who seek treatment, and whose cognitive functioning is affected by a composite of their symptoms.

5. The level of general psychopathology must be considered, especially as a function of the effects of chronicity on cognitive abilities. Predictions here will need to account for cognitive effects of chronicity from trauma acquired as an adult and during development as a child. Chronicity is reflect-

ed separately by DSM Axis I and DSM Axis II disorders and by the interactions between these axes. In addition to possibly altering information-processing abilities, early traumatic experiences may affect the course of cortical development in children in a manner that consequently shapes the development of a trauma-adapted personality, manifesting as BPD and MPD, and other syndromes such as attention deficit disorder and learning disabilities. Emerging findings point to an overlap among these syndromes, suggesting the effects of trauma as the common denominator.

6. Can existing neurological disorders and syndromes be employed as models of anatomical substrates to explain the expression of some PTSD symptoms (e.g., fugue states, reduplicative paramnesias, temporo-limbic epilepsy, Capgras syndrome)?

7. Does clear neurological disease overshadow the psychiatric effects of PTSD, or when comorbidly present, do the two interact to create a clinical hybrid?

8. Can a "pure" PTSD lesion be acquired in the absence of biological predisposition, or does PTSD develop because of preexisting vulnerabilities that might include residual brain effects from earlier traumas or acquired injuries?

Once a theoretical model is specified, practical issues will remain for testing its postulates. Some considerations include the following:

1. Can a representative PTSD group be defined for studying the isolated effects of trauma on cognitive variables that is without pretrauma and posttrauma confounds?

2. Would findings from studying this group be meaningful and generalize to the type of chronic PTSD patient commonly presenting for treatment?

3. What are the best methods for interpreting how much of the neuropsychological test performances are a reflection of PTSD versus the plethora of comorbid psychiatric, medical, and neurological conditions?

4. Are the psychological effects of trauma established sufficiently and the investigation methods advanced enough for use in examining which of the parameters of the traumatic experience predicts patterns of neurocog nitive processing?

5. If early trauma is implicated as a determining factor in shaping neurocognitive functioning, then what factors are most critical for defining control- and target-study groups?

6. If the effects of trauma on neuropsychological functioning are hypothesized to be different for adults than for children and adolescents with developing brains, then what are the most sensitive measures to select for each population—clinical neuropsychological or experimental neuropsychological measures? Very basic knowledge of neuropsychological performances in PTSD populations has yet to be established. At this point in time, the

sensitivity of these instruments for measuring the neurocognitive aspects of PTSD has not been established, nor has it been disconfirmed. It is unclear which test instruments are most sensitive, or if clinical neuropsychological measures are best suited for assessing the cognitive problems accompanying PTSD. Clinical neuropsychological tasks that are useful for examining the effects of neurological disorders on cognitive functioning may prove too prone to ceiling effects in traumatized patients, especially younger adult samples.

7. What are the optimal administration procedures for sampling the neurocognitive effects of PTSD, given available measures? Using tests with established literatures to study PTSD is a reasonable starting strategy, but if future studies that are better controlled find few deficits, then the overall composition of test batteries will need to be reconsidered, and testing procedures may need to be modified. Given that PTSD presents clinically with static and phasic components, the combination of history-collection procedures, tests selected, and the administration methods employed should adequately sample the variation. To increase sensitivity to the cognitive problems reported by patients, research protocols may need to test PTSD patients in affectively primed and unprimed states as an analog that mimics information-processing conditions in everyday settings.

Answers to these questions will dictate study designs, sampling strategies, and the search for methods most sensitive to the corresponding cognitive dysfunctions.

Treatment Integration

Functional recommendations from neuropsychological evaluations will need to he integrated more effectively with clinical treatment protocols. At present, if impairments are detected, typical test findings are less likely to be interpreted from a PTSD framework and more likely summarized as performances showing a diffuse patterns of neurocognitive deficits, ranging in severity from mild to moderate, similar to organic-psychiatric or general-psychiatric groups. As noted previously, some samples, and some individuals within PTSD samples, will exhibit greater degrees of functional impairment in cognitive domains resembling other brain syndromes because of coexisting closed bead injuries, learning disabilities, attention deficit disorder, seizure disorders, medical problems, and substance abuse. In chronic clinical PTSD samples with coexisting mood disorders, the effects of depression will likely be evident in the test findings as well.

To increase the utility of the testing process and the findings obtained, results need to be more closely linked to clinical behavioral problems experienced by the patient. For instance, do PTSD patients with anger-con-

trol problems and rage episodes demonstrate patterns of deficits on tests that are affected by frontal lobe dysfunction? Are scanning and tracking errors on testing correlated with clinical hypervigilance, or does the presence of hypervigilance result in better performances on these tests? Do the type of amnestic problems defined for neurological disorders manifest similarly on testing for PTSD patients with psychogenic amnesia? Can levels of PTSD symptom severity be explained by decreased overall cognitive capacity as demonstrated across a battery of tests requiring sustained mental activity? Answering these questions would add to practical impact of the testing results.

Although greater integration of neuropsychological assessment can occur in the future, current application can reap immediate benefits. Presently, in many settings, PTSD patients may be placed into treatment protocols that implicitly require certain levels of sustained attention and concentration focus, plus minimum capacities for verbal abstraction and memory (e.g., various forms of group and process-oriented individual therapy). Neuropsychological test findings can assist in matching patients to treatment with greater accuracy than a simple mental status examination. Neuropsychological testing can also be used when explaining to patients that portions of their reactions and internal experiences may be a product of brain injury. For example, a veteran undergoing an evaluation reported being overwhelmed with guilt feeling surrounding the responsibility he felt for the death of close friends in a combat firefight. He also experienced **30** days of coma from a closed head injury and subsequently began hearing auditory hallucinations of his friends' voices, telling him that he was at fault for their deaths. He concluded that he caused the voices as a way to punish himself for surviving the battle when they had died; the combination of hallucinations and attributions invariably produced episodes of deep depression and suicidal ideation. In this case,in which anatomical lesions were present and documented in the medical record (e.g., diffuse abnormalities on the MRI scan from the previous closed head injury), feedback about the effects of brain damage as a possible cause of the voices was very useful in modifying his attribution that they were self-caused as punishment. This information, combined with his neuropsychological findings, provided the foundation of an alternative explanation for his symptoms and subsequently reduced for him the sustained discomfort that typically had continued after each hallucinatory episode.

Observing the PTSD patient's overall response to a battery of tests provides additional clinical benefit. The amount of effort required by the patient to sustain concentration and complete a neuropsychological test battery can inform clinicians about patient fatigability and the likely emergence or change in symptoms resulting from sustained mental exertion. As the previous section on reactivity to the testing process showed, patients can exhibit reactions during testing that are not necessarily elicited during the inter-

view process. These reactions during structured testing may be analogous to the types of responses that develop when faced with daily challenges.

In conclusion, state-of-the-art neuropsychological assessment of PTSD relies mainly on methods developed in previous decades, before the official PTSD diagnosis was formalized in the DSM-III. The adequacy of conceptual models and testing methods needs to evaluated to determine appropriateness for examining cognitive dysfunction in PTSD. Expanded application of new imaging techniques and the introduction of computer-based testing protocols will augment the available non-self-report methods for examining PTSD cognitive processes. Advances in our understanding of the effects on neurocognitive functioning resulting from traumatic experiences will occur in conjunction with ongoing theorizing and the many parallel lines of investigation from all sectors of trauma study. Although trauma investigators with diverse perspectives on causality may depend upon levels of analysis that assume different degrees of parsimony, each contributes a complementary piece to the conceptual puzzle. Future developments will continue to define the role of neuropsychological approaches as the intermediary between the psychological and neurobiological investigations of trauma.

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